Authors' reply: We thank Dr Rao for his comments. We are sorry that it is unclear to Dr Rao as to precisely how the location of infarcts influenced the quantification of vascular pathology in this study. We would refer him to the discussion of neuropathological prevalence of dementia subtypes in which we hoped it was clearly stated that "the increased prevalence of infarction is more likely to reflect the stress laid on recording the presence of all infarctions regardless of size or location". Thus, when assessing the validity of these criteria no heed was taken as to whether they were to be considered as strategic or otherwise. Periventricular white matter lesions were not a prominent feature of these cases.

That he finds the low sensitivity of the NINDS–AIREN criteria to be striking is, of course, one of the main points of the paper but one that has been shown elsewhere and referenced in the paper (Gold *et al*, 1997).

The concept of a "nosologically heterogeneous group of disease processes" is precisely the concept that is at fault in dementia research, and one which impedes progress into looking for common causation. Indeed, we can do no better than also quoting Prince, but from a later paper, "It is perhaps regrettable that the clinical distinction between VAD and AD has become so strongly established in advance of clear population-based epidemiological evidence for the existence of distinct disorders with discrete aetiologies" (Prince, 1995).

Gold, G., Giannakopoulos, P., Montes-Paixao, C., et al (1997) Sensitivity and specificity of newly proposed clinical criteria for possible vascular dementia. *Neurology*, **49**, 690–694.

Prince, M. (1995) Vascular risk factors and atherosclerosis as risk factors for cognitive decline and dementia. *Journal of Psychosomatic Research*, **39**, 525–530.

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Neurodevelopmental delay in the severely ill

Sir: Sigurdsson *et al* (1999) conclude that subjects with severe early-onset mood disorders have a lower IQ than those with mild or moderate forms of juvenile-onset affective illness, and present this finding as evidence supporting their hypothesis of neurodevelopmental delay in the severely ill group. We argue that their data do not support this conclusion.

Their study shows that a greater percentage of cases of severe early-onset affective disorder have an IQ below 85 compared with a control group of equally early-onset but milder, non-psychotic depression. For both cases and controls, Wechsler Intelligence Scale for Children (WISC (Wechsler, 1949), or its revision, WISC–R (Wechsler, 1974)) IQ data are available for only a minority of subjects (42%). For the majority, the IQ estimate was taken from the original clinical assessment that took account of "all the available information". This is the non-standardised impression of various clinicians over an 18-year period. The authors present no correlation between measured IQ and estimated IQ, despite these data being available. There is, therefore, no measure of the validity of the estimated scores. Indeed, the authors note that low intelligence and learning difficulties were mentioned infrequently in referral letters and assessment summaries, which suggests that finding an IQ of less than 85 was unexpected in some of the subjects in which it was found. This also raises doubt about the validity of the estimated IQs. Further analysis of IQ is based only on the limited sample in whom formal WISC IQs are available. There is no attempt to assess whether this limited sample is representative of the cases and controls as a whole.

The authors give no clear account of the timing of IQ assessment, but imply that it was carried out during the index episode. This is a major potential confounder as IQ may be influenced by ongoing symptoms (for example, Sackeim *et al*, 1992); lower IQs in the more severely ill group could be a consequence of illness rather than an antecedent. To test the hypothesis a measurement of premorbid IQ is essential.

The assumption that IQ acts as a proxy measure for neurodevelopmental impairment can be challenged. Performance on intelligence tests is influenced by heredity, antenatal and infant nutrition (Lucas *et al*, 1992), various socio-economic factors (Elliott, 1988), gender (Hedges & Nowell, 1995) and possibly ethnicity (Elliott, 1988). The cases and controls differed significantly in their parental country of origin, a measure of ethnicity, and the gender differences almost reached statistical significance.

Sigurdsson *et al* (1999) conclude that neurodevelopmental antecedents are overrepresented in severe early-onset mood disorder and that this is supported by contemporaneous IQ data. We argue that such use of incomplete measures of IQ is invalid. The use of unsatisfactory data to support their conclusions is unsafe.

Elliott, R. (1988) Tests, abilities, race and conflict. *Intelligence*, **12**, 333–350.

Hedges, L. V. & Nowell, A. (1995) Sex differences in mental test scores, variability, and numbers of high-scoring individuals. *Science*, **269**, 41–45.

Lucas, A., Morley, R., Cole, T. J., et al (1992) Breast milk and subsequent intelligence quotient in children born preterm. *Lancet*, **339**, 261–264.

Sackeim, H. A., Freeman, J., McElhiney, M., et al (1992) Effects of major depression on estimate of intelligence. *Journal of Clinical and Experimental Neuropsychology*, **14**, 268–288.

Sigurdsson, E., Fombonne, E. Sayal, K., et al (1999) Neurodevelopmental antecedents of early-onset bipolar affective disorder. *British Journal of Psychiatry*, **174**, 121–127.

Wechsler, D. (1949) *Manual for the Wechsler Intelligence Scale for Children*. New York: Psychological Corporation.

— **(1974)** *Wechsler Intelligence Scale for Children – Revised*. New York: Psychological Corporation.

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Authors' reply: We are glad to be able to report in more detail the IQ findings from our study while responding to the points raised above. Three main issues need to be addressed. First, whether not having IQ test scores for all subjects (available for 42% of cases and 42% of controls) made it unsafe to use these scores to validate our main findings; second, the interpretation of the differences observed in IQ scores between the groups; and finally, whether it is appropriate to use IQ test scores as a proxy measure of neurodevelopmental antecedents in our sample.

With regard to the first issue, cases and controls for whom IQ test scores were available did not differ significantly from other cases and controls, respectively, with regard to gender, social class or parental country of birth. They were, however, slightly younger, on average, at the onset of the index episode. The magnitude of the age difference was similar but reached statistical significance only for the cases (cases 13.6 *v.* 15.0 years, P=0.019; controls 13.8 *v.* 14.6 years, P=0.23, Student's t-test). Although the

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gender ratio between cases and controls was unequal, albeit non-significantly so, this could not account for the marked differences observed in IQ scores. On the one hand, the difference between the two groups was around one standard deviation, which is much more than the commonly observed difference of 0 to 1 points between genders (Hedges & Nowell, 1995). This was the case both for verbal (difference=12.5 points, P=0.01, Student's t-test) and performance IQ (difference=19 points, P=0.002, Student's t-test). On the other hand, males had a higher mean IQ (WISC or WISC-R) score, and this was observed both for cases and controls, and there were more males among the cases but more females among the controls. The mean IQ scores in the sample were 97.1 for females, 98.9 for males (difference=1.8, P=0.75, Student's t-test, cases: 85.0 females v. 90.1 males, P=0.60; controls: 104.5 females v. 107.5 males, P=0.64).

With respect to the second main issue, it has been routine practice at the Maudsley to defer IQ testing of psychotic or very depressed adolescents and those on high doses of antipsychotic medication. The performance IQ may yet not adequately reflect premorbid IQ. However, the verbal IQ was also significantly lower for cases than controls (cases: 92.6, s.d. 15.9; controls: 105.1, s.d. 13.1, P=0.01, Student's t-test). Scores that were regarded as unreliable by the assessing psychologist were not included in the study. On a few occasions more than one IQ test had been performed on the same subject in adolescence, in which case only the highest scores were entered into the database for statistical analysis.

Subjects for whom psychometric results were not available had IQ estimates imputed by clinicians contemporaneously, as this was and remains an item in the 'item sheets' which teams are expected to complete for every patient. This is usually estimated in light of school reports and grades, history given by informants and interviews with the subjects, the main purpose being to identify those with very low IQ. We only used these estimates to create a binary variable with a cut-off of 85 for logistic regression modelling. In the total sample, eight subjects (all cases) had an estimated IQ below 85. The validity of this categorisation had been confirmed by formal psychometric testing.

Lastly, 14 out of those 15 patients in the sample who had experienced a neurodevelopmental delay (11/12 cases, 3/3 controls) had their IQ tested. The mean IQ scores differed significantly between these subjects and those who had not experienced neurodevelopmental antecedents (88.6 v. 104.3, P=0.005, Student's t-test). This relationship provided strong support for our conclusion, linking IQ test scores to developmental delay. As referred to by McConville & Walker, other factors may also have contributed to the discrepancy in mean IQ between the groups. We stand by our conclusion that the differences in IQ scores between cases and controls support our main results, the significant association between neurodevelopmental antecedents and early-onset bipolar affective disorder in our sample.

Hedges, L. F. & Nowell, A. (1995) Sex differences in mental test scores, variability, and numbers of high-scoring individuals. *Science*, **269**, 41–45.

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Homicides and community care: the evidence

Sir: Like Taylor & Gunn (1999), we have been looking at statistics bearing on homicides and serious violence by mentally ill persons. We also see no support for an alleged failure of community care.

We have concentrated on the past decade. Between 1987 and 1995 the number of National Health Service (NHS) beds in England and Wales continued to fall (67 130 to 39 480), compensated in number but not in staffing levels by beds in the private sector and in staffed residential homes. As Taylor & Gunn point out, while total homicide convictions in England and Wales increased substantially after 1965, this was not paralleled by Section 2 (diminished responsibility) manslaughter convictions under the Homicide Act 1957. Indeed, we calculate they declined significantly in the decade 1987–1997 (regression slope on year: $B=-2.94$, s.e.=0.60; $t=-4.9$; $P>0.001$).

However, while homicides by people with mental illness have been steady or declined, the number of compulsory orders under the Mental Health Act 1983 (MHA) has increased dramatically (Fig. 1). Department of Health statistical bulletins (Department of Health, 1996, 1997, 1998*a*,*b*) show a 64% increase in compulsory admissions in England (1986–1996),

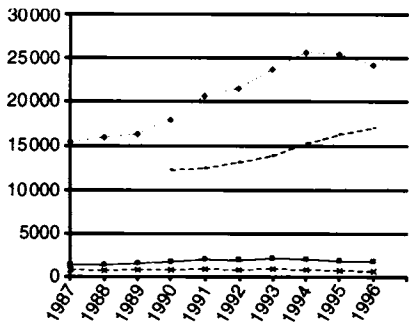

Fig. 1 Compulsory admissons to NHS facilities, special hospitals and private mental nursing homes in England: 1987–1996. Total orders (- -◆- -), changes from informal to compulsory order (- - -) and total court and prison orders (–■–) and Section 37 hospital orders (- ✱ -).

and a 40% increase in use of MHA sections following initial informal admission (1990–1996). However, total court and prison orders have not changed over the period 1987–1996, despite a marked increase in pre-sentence Section 48 transfers from prisons to hospitals as part of a policy aimed at providing more timely psychiatric care for remanded prisoners with mental illness. Especially noteworthy is the fact that Section 37 hospital orders (with or without restrictions) made after conviction have remained unchanged.

Perhaps the emphasis on the tenets of the Care Programme Approach (CPA) and implementing assertive community treatment have resulted in both an increase in MHA sections and a reduced risk of homicides. Of special concern is the possibility that widespread fears that one of their patients may kill have lowered mental health teams' thresholds for using compulsion, possibly quite unnecessarily, and at substantial cost to patients' liberties.

Despite the pronouncements of homicide inquiries, sadly biased by only examining worst outcomes, community psychiatric services have probably implemented the major elements of the CPA and are more assertive than ever before in ensuring that patients do not fall through the net of services. A national survey conducted by us for the Department of Health has shown that about 1% of the population is on a CPA register, and about 1% of these are on a Supervision Register (Bindman *et al*, 1998). Ironically, it is plausible that by being more accessible and reaching out more effectively to those suffering from a mental disorder who would have been

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